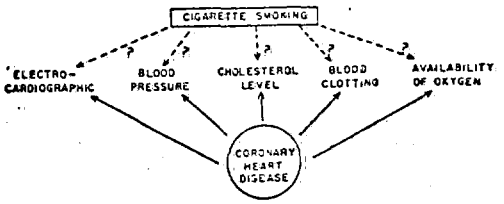
VIII. PREVENTABLE RISK FACTORS PROBABLY INFLUENCED BY SMOKING



XIII PREVENTABLE RISK FACTORS (INFLUENCED BY SMOKING)

A. Electrocardiogram and Myocardial Status---------- 201
B. Hypertension---------- 204
C. Hypercholesterolemia--------- 207
D. Blood Coagulation---------- 214
E. Availability of Oxygen and Associated Changes in the Blood---------- 219

The preventable risk factors discussed in this section are those that are believed to be exaggerated by smoking. This supposition is based on observations of the effects of acute exposure to smoking in humans and animals and these are discussed in Section IX. The differences between habitual smokers and nonsmokers are described below. It should be stated at the outset that there is no concrete evidence for the hypothesis that chronic smokers show exaggerated electrocardiographic signs of ischemia, develop hypertension, hypercholesterolemia and hypercoagulation of the blood. The problem of reduced availability of oxygen in the blood of the habitual smoker is a complex one and is discussed under carbon monoxide (Section XI).

The general articles relating to the preventable risk factors are contained in the following Additional Bibliographic Lists:

List No. 19. Investigation of preventable risk factors in foreign countries.

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List No. 20. Ischemic changes in the electrocardiogram.
List No. 21. Hypertension as a risk factor.
List No. 22. Cholesterol and coronary heart disease.
List No. 23. Thrombosis induction and prevention.

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THROMBOSIS -- INDUCTION AND PREVENTION

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VIII-A. Electrocardiogram and Myocardial Status

The electrocardiogram has been used to assess the adequacy of oxygenation of the myocardium. The signs of ischemia in the electrocardiogram include depression of the S-T segment, inversion of the T wave and alteration in the QRS complex. These are useful in the diagnosis of coronary heart disease and in the detection of coronary-prone individuals (see Additional Bibliographic List No. 20).

The electrocardiogram of smokers, in contrast to that of nonsmokers, has been examined by several groups of investigators. Patients who develop chest pain during smoking show ischemic signs characteristic of tobacco angina (Boas and Levy, 1936; Wilson and Johnston, 1941; see also Section I-A). In patients prior to developing infarction as well as in those who have already developed it, there is a predominance of smokers over nonsmokers (Lafrenz, 1970; Iglesias Leal et al., 1971; Zdichynec and Prokš, 1971). This does not necessarily imply a cause and effect relationship because there are other risk factors which produce infarction.

The examination of the electrocardiogram of healthy smokers failed to show a significant difference in frequency of the ischemic pattern in the electrocardiogram as compared with that of nonsmokers (Agnese and Buonvino, 1964; Gelin et al., 1967; van Buchem, 1970; Hagerup, 1971). In similar surveys by other investigators, there were more smokers in the group that showed ischemic response in the electrocardiogram during exercise. Doyle and Kinch (1970) reported that 69% of subjects giving an

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ischemic response were smokers, whereas 37% of those with a normal electrocardiogram were smokers. Kattus et al. (1971) obtained the respective incidence of 59% and 42%. As in other studies, the role of various risk factors could not be excluded. When the heart was stressed by hypoxia, there was no difference between smokers and nonsmokers in the T-area vectors of the electrocardiogram (Abel, 1971). The QRS complex showed a right axis deviation in some smokers (Borden and Ibrahim, 1970). The polarcardiogram, which graphs the polar coordinates of the heart vectors vs. time, indicate a difference between smokers and nonsmokers (Dower et al., 1972). The complexity of the technique prevents its widespread application to determine the significance of the difference.

The heart of smokers in contrast to that of nonsmokers has been investigated by means of special techniques. The ballistocardiogram and other techniques for cardiac output measurement do not show any difference between smokers and nonsmokers in this respect (Starr (1954) and also Section IX-A).

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VIII-B. Hypertension

Hypertension is an important risk factor in coronary heart disease. The epidemiologic investigations of Pell (1961), Oberman et al. (1969) and Kannel et al. (1969, 1971) in the United States, of Tibblin (1967) in Sweden and of Kahn et al. (1972) in Israel demonstrate the major role of hypertension in the pathogenesis of coronary heart disease. There are other studies outlined in Table VII-E.

Essential hypertension as a separate disease entity has a set of precursors which include age, sex, familial, psychologic and dietary factors (Thomas, 1958; Lowenstein, 1961; Thomas et al., 1964; Paul and Ostfeld, 1965; Paul, 1971). Smoking is not one of them because smokers in the general population have a lower mean blood pressure than nonsmokers (Edwards et al., 1959; Borhani and Hechter, 1964; Clark et al., 1967). In groups preselected for absence of hypertension, there is no difference in mean blood pressure but a slight diminution in the response to the cold pressor test (Blackburn et al., 1960). A reduction of sympathetic vascular tone which occurs in smokers cannot be considered as contributing to a rise in blood pressure.

When essential hypertension appears together with coronary heart disease, the form of therapy indicated is to reduce the blood pressure by drugs (see Additional Bibliographic List No. 21). The influence of cigarette smoking in such a situation has not been investigated, since most studies relate either to essential hypertension of to coronary heart disease but not to both.

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VIII-C. Hypercholesterolemia

Hypercholesterolemia is one of the risk factors for coronary heart disease. In prospective studies, most patients who develop this disease also show an elevation of cholesterol and triglyceride levels in the blood serum (Kannel et al., 1971). Hypercholesterolemia is associated with other risk factors, including emotional stress, behavioral pattern and diet (Bronte-Stewart et al., 1955; Wertlake et al., 1958; Rosenman and Friedman, 1963; Rosenman et al., 1964; Jenkins et al., 1968; Pincherle, 1971). Therapy of coronary heart disease patient includes the use of diet and hypocholesterolemic drugs with the expectation that this would improve the prognosis in these cases (Cohen, 1969; Hughes, 1970; Coronary Drug Research Project, 1970; Newcastle-upon-Tyne Study Group (Dewar), 1971; Dayton, 1971; Dewar, 1972). The level of cholesterol drops following myocardial infarction per se, so that it is not reasonable to depend on this measurement to assess the severity and recovery from the infarction(Watson et al., 1963; Kurien and Oliver, 1966; Soloff and Schwartz, 1966).

The influence of cigarette smoking on the cholesterol level blood serum has not been completely settled. The results of 26 investigations are summarized in Table VIII-C. In half of these studies, smoking is associated with slight elevation of the cholesterol level, but in the other half there is no such elevation. When healthy subjects are matched according to their age group, body weight and behavioral pattern, there is no significant difference between smokers and nonsmokers (Schwartz et al., 1971).

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In patients with coronary heart disease, those who smoke have higher levels of cholesterol than nonsmokers and the arteriogram shows obstructive lesions more frequently in the former (Cramer et al., 1966). Smoking cannot be regarded as the cause of hypercholesterolemia and obstructive lesions, since other risk factors have not been excluded.

(Table VIII-C appears on the next two pages.)

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TABLE VIII-C.

Role of cigarette smoking in subjects with hypercholesterolemia

Country/Investigators	Subjects	Role of Cigarette Smoking
United States		
Gofman et al., 1955	123 healthy	Elevation of lipoproteins and cholesterol.
Burney and Enslein 1972	222 healthy	No difference in 5 year level of cholesterol compared to nonsmokers.
Spain and Nathan 1961	3,000 healthy males	Heavy smoking associated with higher cholesterol levels.
Talbott 1964	40 healthy males	Smoking while on a high fat diet suppressed rise in cholesterol and total artificial fatty acid.
Australia		
Mundy and Cutforth 1972	80 patients with myocardial infarction	Hyperlipoproteinenmia not associated with smoking.
Belgium		
Kornitzer et al., 1971	539 subjects	Hyperlipoproteinemia associated with coronary heart disease but not with smoking.
van Houte and Kesteloot 1972	42,804 males	Heavy smoking associated with higher cholesterol levels.
Finland		
Konttinen 1962	314 healthy young men	Smoking not related to cholesterol and phospholipids in serum.
Hernberg 1964	862 businessmen	Smoking not related to cholesterol level or capacity for physical work.
France		
Richard et al. 1969	7,672 subjects	Weak link between tobacco and cholesterol level and triglyceride
Lellouch and Beaumont 1967	1,053	Smoking not related to cholesterol level if body weight and age considered
Germany		
Gross [illegible], 1970	38 subjects	Smoking not related to cholesterol levels.
Great Britain		
Acheson and Jessop 1961	221 elderly men	Smoking not related with cholesterol or lipoprotein level.
McDonald et al., 1965	976 air force men	Smoking not related with cholesterol level.
Pincherle and Wright 1967	2,000 businessmen	Heavy smoking associated with higher cholesterol level.
Pozner and Billimoria 1970	64 young subjects	Heavy smoking associated with higher cholesterol and lipoprotein levels.
Pincherle 1971	7,000 businessmen	Heavy smoking associated with higher cholesterol level.
India		
Gupta 1964	286 subjects	Heavy smoking associated with higher cholesterol level.

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Italy		
Mattioli and Mancini 1959	51 subjects	Smoking not related to cholesterol level.
Balestra and Canepa 1967	47 normal and 36 patients	Smoking not related to cholesterol level.
New Zealand		
Smyth et al., 1970	70 patients with coronary heart disease	Smoking habit not reported
Norway		
Romslo 1971	324 military men	Smoking associated with higher triglyceride level.
Spain		
Portugal Alvarez et al., 1972	30 subjects with coronary heart disease	Smoking associated with high free fatty acid level.
Vlaicu et al., 1971	100 patients with coronary heart disease	Smoking associated with higher cholesterol level.
Sweden		
Carlson and Böttiger 1972	3,168 men	Elevated triglyceride and cholesterol observed for coronary heart disease but relation to smoking not reported
Switzerland		
Heyden-Stucky 1965	76 men with myocardial infarction	Smoking and hypercholesterolemia prevalent

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C. Hypercholesterolemia

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VIII-D. Blood Coagulation

The basic process of myocardial infarction consists of development of thrombosis in the atherosclerotic coronary artery. The process of blood coagulation has been investigated in such cases and measurements indicate hypercoagulability of the blood in patients with coronary heart disease (see Additional Bibliographic List No. 22).

Cigarette smoking has long been suspected of increasing coagulability of the blood. Most of the experiments have been carried out on animals and helathy subjects and the observations were made after a brief period of exposure to cigarette smoke (see Section IX-D). The present section pertains to differences between nonsmokers and smokers, which are as follows:

1. Coagulation time. Kedra and Korolko (1966) compared coagulation time in a group of 50 smokers and 50 nonsmokers. There were no significant differences in the various tests performed on the two groups. After the smokers consumed 5 cigarettes, there was a statistically significant shortening of clotting time, of plasma calcium time, and of factor V values, compared with the presmoking values. It is surprising to note that this is the only comparison between smokers and nonsmokers who are healthy. There is no comparison of subjects with coronary heart disease. Patients with ischemic heart disease show a decrease in coagulation time after smoking but there was no comparison with nonsmokers (Orlova, 1971). In an epidemiologic investigation of coronary heart disease, made in India,

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no relation was found between smoking habits, clotting time and incidence of coronary heart disease (Malhotra, 1968).

2. Fibrinogen level. In an examination of 788 men 54 years of age, smokers were found to have a higher fibrinogen level than nonsmokers (Korsan-Bengsten et al., 1972). In young adults, the fibrinogen level was not different as between smokers and nonsmokers (Ogston et al., 1970). The plasma fibrinogen level was not altered acutely by smoking one cigarette. The authors concluded that habitual use of cigarettes over a long period may induce hyperfibrinogenemia.

3. Fibrinolytic activity. The observations relating to fibrinolytic activity and smoking are limited to healthy subjects. Gibelli et al., (1964) reported a reduction in fibrinolytic activity in smokers. Loncin et al., (1968) reported that patients with coronary heart disease showed lower fibrinolytic activity than controls, but they did not investigate the influence of smoking.

4. Platelet aggregation. Although a considerable amount of work has been done to investigate the effect of smoking on platelet function, there is only one such study that can qualify as comparing smokers with nonsmokers. Hawkins (1972) examined three groups: nonsmokers, light smokers (using less than 1 pack a day) and heavy smokers. Changes in platelet activity could be separated into those apparent only after smoking and those detectable in the heavy smoker even after a nonsmoking period. The biphasic platelet electrophoretic mobility change was altered only after

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smoking and had returned to normal after 15 minutes. On the other hand, statiscally significant changes in thromboelastograph measurements were apparent in heavy smokers even during a nonsmoking period. Both acute and chronic changes seemed to result in an increase in platelet aggregation in the heavy smoker.

5. Influence of oral contraceptives. Frederiksen and Ravenholt (1970) suggested that the use of oral contraceptives and heavy cigarette smoking interact synergistically to increase the likelihood of thromboembolic disease. They reached this conclusion by a reanalysis of the data of Vessey and Doll (1968) who doubted that such an association existed. The interpretation by Vessey and Doll was supported by the previous study of Sartwell et al. (1969), in which there was no correlation between incidence of thromboembolism and the amount of cigarettes consumed. Sartwell (1971) has reexamined the literature and his own material and has concluded that, although there was some association of smoking with recent use of oral contraceptives, evidence was still lacking that smoking enhanced their effect in producing clotting.

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The interaction between contraceptives and cigarette smoking can be resolved by measuring the coagulability of blood. The effect of contraceptives on blood coagulation has been examined by Brakman et al. (1967) but these observations have not been extended to a comparison of smokers and nonsmokers. Patients who developed myocardial infarction in the course of using oral contraceptives have been examined thoroughly for the presence

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of risk factors by Oliver (1970) although he failed to measure blood coagulation. When these deficiencies are corrected, the potential hazard of combining smoking and the use of contraceptives will be more clearly defined.

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VIII-E. Availability of Oxygen and Associated Changes in the Blood

In recent years, there have been claims that the carbon monoxide contained in cigarette smoke reduces the availability of oxygen in the blood. The formation of carboxyhemoglobin and its consequences are discussed in Section XI.

There are other changes in the blood of smokers which differentiate them from nonsmokers. They are as follows:

1. Oxygen-dissociation curve. Aside from the formation of carboxyhemoglobin, there is a change in the affinity of hemoglobin for oxygen in the blood of smokers. This change is discussed in Section XI. Metcalfe et al. (1969) reported that patients with coronary heart disease and low output failure show a decreased affinity of the blood for oxygen. This is considered a compensatory adjustment to poor tissue blood flow, promoting the diffusion of oxygen from blood in tissue capillaries to intracellular sites of utilization. The status of this adjustment has not been examined in smokers who develop low output failure.

2. Hematocrit. There is an increase of the hematocrit in patients who develop acute myocardial infarction (Burch and De Pasquale, 1962; Stables et al., 1967). The resulting increase in the viscosity of the blood influences the free flow of blood and has been postulated to cause infarction (Dintenfass, 1969). This theory does not apply to smokers because there is no increase of the hematocrit in the blood.

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3. Blood cells. The distribution of blood groups is different between smokers and nonsmokers. With reference to white males, Cohen and Thomas (1962) reported that there appeared to be a significant deficiency of group B individuals among heavy smokers and an excess of group B persons among nonsmokers. There was also an excess of Rh negative individuals among the white occasional smokers. There were no significant differences among the Negro males. A genetic difference in blood groups has also been reported by Jick et al. (1969) in patients who develop coronary heart disease. The subsequent correspondence with Nefzger et al. (1969) and that with Havlik et al. (1969) indicate the problems in arriving at an unequivocal interpretation of prospective studies.

Another feature reported as appearing in smokers and patients with myocardial infarction is the occurrence of eosinophilia and lymphocytosis (Zdichyriec, 1971). This has been interpreted as an allergic response to tobacco but may also be a genetic response.

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